AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 108:311–319 (1999)

A Prehistoric Example of Polydactyly From the Iron Age Site of Simbusenga, Zambia

KIMMARIE A. MURPHY*
Department of Anthropology, Indiana University, Bloomington, Indiana 47405

KEY WORDS paleopathology; preaxial; pedal; Africa

ABSTRACT Human burials, dated AD 1100–1500, were examined from the Iron Age site of Simbusenga, located some 35 miles northwest of Victoria Falls in Zambia. Pedal polydactyly was discovered in the fragmentary remains of a young adult of indeterminate sex aged 14–25. The preaxial form of polydactyly is indicated with bilateral involvement of the first metatarsals. There is incomplete hypoplastic duplication of both first metatarsals with broad heads for the metatarsal-phalangeal joints. No digital malformations were found in the other seven individuals with feet and/or hands from the site. Several studies point to autosomal dominance for cases of isolated polydactyly, but inheritance and patterning of preaxial polydactyly are still incompletely understood. The condition is also found in conjunction with genetic malformation syndromes such as Acrocephalypolysyndactyly, Lambotte, Orofacio-digital, and VATER. High frequencies of polydactyly are reported for African and African-American populations, but further analysis reveals that the bulk of previously reported cases of polydactyly are representative of the postaxial form as opposed to the preaxial expression seen here. Am J Phys Anthropol 108:311–319, 1999.

Polydactyly, the congenital duplication of one or more digits, is one of the most common malformations in humans today (Woolf and Myrianthopoulos, 1973; Christensen et al., 1981). Population studies in the US (Sesgin and Stark, 1961; Chung and Myrianthopoulos, 1968) rank polydactyly among the ten most common congenital birth defects, which also include clubfoot, hydrocele, hypospadias, cryptorchidism, cardiovascular defects, cleft palate, harelip, syndactyly, and central nervous system anomalies. The frequency of polydactyly varies widely among populations. For example, the incidence of polydactyly per 1,000 births in Utah (Woolf and Woolf, 1970) is 0.47, in Uruguay, Chile, and Argentina the incidence is 1.01 (Castilla et al., 1973), and in Nigerian students it is as high as 22.78 (Scott-Emuakpor and Madueke, 1976). Researchers have often pointed out the high frequency of polydactyly in populations of African vs. European ancestry. Frazier's (1960) study of consecutive live births in Baltimore found an incidence per 1,000 births of 3.6 for African-Americans vs. 0.30 for Euro-Americans. Similar results were noted by Sesgin and Stark (1961), who found a ratio of 2:1 in African-Americans vs. Euro-Americans. Finally, Woolf and Myrianthopoulos (1973) reported that the incidence for African-Americans per 1,000 births was 13.7 vs. 1.3 for Euro-Americans.

References to polydactyly exist within the mythology of various cultures; for example, Celtic mythology depicts an Irish hero with seven fingers and toes (Cross, 1952). Nicolai

Grant sponsor: Fulbright.

*Correspondence to: Kimmarie A. Murphy, Dept. Anthropology, SB 130, Indiana University, Bloomington, IN 47405. E-mail: kiamurph@indiana.edu

Received 27 January 1998; accepted 12 November 1998.

and Schoch (1986) made reference to the six-fingered, six-toed giant slain by David's brother in the Gath war from II Samuel 21:20 of the Bible. Historical sources indicate that Anne Boleyn also had some form of polydactyly (Weir, 1991). However, despite these historic examples, prehistoric evidence for polydactyly has been sparse. To the best of my knowledge, only Barnes (1994) and Hill and Case (1996) have identified polydactyly in prehistoric human remains, and their examples were of postaxial pedal polydactyly. Rock art from the American southwest (for a review of rock art literature see Barnes, 1994) and Argentina (AD 1000) also attest to the antiquity of ideas about polydactyly if not the biological condition itself (Castilla et al., 1973).

CLINICAL CLASSIFICATION OF POLYDACTYLY

Polydactylous manifestations are described according to their anatomical location on the proximal, intermediate, or distal segments of the hand/foot (Temtamy and McKusick, 1969; Venn-Watson, 1976; Phelps and Grogan, 1985; Watanabe et al., 1992). Digital duplications range from boneless soft tissue structures (i.e., pendiculated postminimus) to incomplete or complete bony duplications (Christensen et al., 1981). Preaxial duplications occur on the first digit or radial/tibial side of the second digit, while postaxial duplications are located on the ulnar/fibular side of the second digit and either side of digits three through five. Combinations of preaxial or postaxial polydactyly on hands and feet are referred to as mixed (Christensen et al., 1981).

Temtamy and McKusick (1978) described five basic types of preaxial polydactyly: type 1, MT I polydactyly; type 2, triphalangeal; type 3, MT II polydactyly; type 4, polysyndactyly; and type 5, MT I polydactyly with tibial defects. Burial 5's polydactyly (in this study) is classified as type 1. Since Burial 5 exhibits polydactyly of the first metatarsals, this study will deal only with those polydactylies affecting the first metatarsals. Type 1 preaxial polydactyly has been further subdivided based on morphological variations (Venn-Watson, 1976; Phelps and Grogan, 1985; Watanabe et al., 1992) and includes the following categories: 1) short, block MT I, 2) wide head MT I, 3) MT I with hypoplastic lateral member, 4) MT I with hypoplastic medial member, and 5) complete duplicated MT1 (Fig. 1).

ETIOLOGY

The exact human embryological parameters behind polydactyly are unknown. However, experiments on rat embryos produced preaxial polydactyly when cells in the limb bud mesoderm layer were killed or disrupted. The disruption led to excess tissue formation on the apical portion of the ectodermal ridge with resulting supernumerary digital development (Scott et al., 1980; Phelps and Grogan, 1985; Graham et al., 1981).

Most preaxial polydactyly occurs as an isolated genetic malformation with both autosomal dominant and recessive inheritance patterns and varying degrees of penetrance depending on the type of polydactyly (Sonoga and Guttmann, 1989; Woolf and Woolf, 1970; Radhakrishna et al., 1993; Phelps and Grogan, 1985; Walker, 1961). However, preaxial polydactyly tends to be autosomal dominant (Phelps and Grogan, 1985; Dooley and Niehaus, 1985). Of the three types of polydactyly (preaxial, postaxial, and mixed), postaxial polydactyly occurs most frequently, although there is some population variation in expression (Woolf and Myrianthopoulos, 1973; Watanabe et al., 1992). Watanabe et al.'s (1992) study of pedal polydactyly in Japan looked at 265 individuals with polydactyly, 8% (n = 22) of which had preaxial polydactyly. Of the 22 patients with preaxial polydactyly, the duplications were bilateral in 14 individuals. Venn-Watson's (1976) study of pedal polydactyly in New Mexico found bilateral involvement in over 50% of the patients with polydactyly.

Mixed and postaxial polydactylies are more frequently associated with genetic syndromes than preaxial types (Dooley and Niehaus, 1985; Phelps and Grogan, 1985). Genetic syndromes associated with pedal preaxial polydactyly can be found in Table 1. This list of syndromes does not include the syndromes associated with triphalangeal thumb (Klippel-Feil and Holt-Oram) or those syndromes that are lethal in nature (hydro-

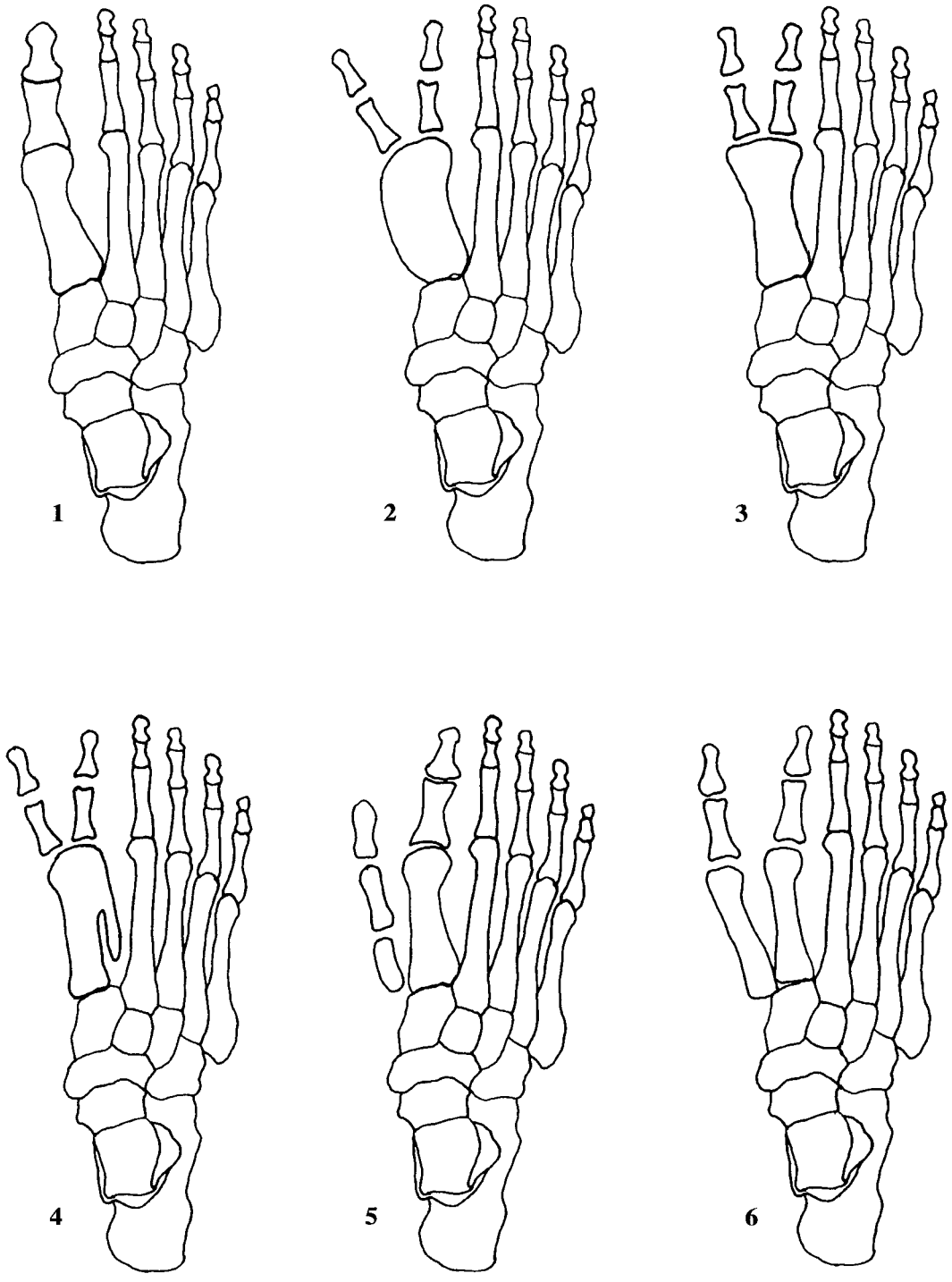

Fig. 1. Morphological variants of preaxial polydactyly in MT I. 1, normal MT I; 2, short, block MT I; 3, wide head MT I; 4, MT I with hypoplastic lateral member; 5, MT I with hypoplastic medial member; 6; complete duplication of MT I. (Based on Watanabe et al., 1992 and Masada et al., 1987.)

TABLE 1. Syndromes associated with pedal preaxial polydactyly

Syndrome	Inheritance[1]	Additional skeletal malformations
Acrocephalypolysyndactyly[2]		
Type I: Noack Syndrome	Dominant	Enlarged thumbs
Type II: Carpenter's Syndrome	Recessive	Acrocephaly, craniosynostosis, shallow orbits, brachydactyly,
Type III: Sakati Syndrome	Dominant	Bowed femurs, hypoplastic tibiae, deformed fibulae
Acrocallosal Syndrome[3]	Recessive	Postaxial polydactyly, macrocephaly, bipartite clavicle
Curry-Jones Syndrome[3]	Unknown	Unilateral craniosynostosis, preaxial polydactyly of hands
Ectodermal Dysplasia Syndrome	Dominant	Thin dental enamel
Goiter, multinodular, cystic renal disease, and digital anomalies[3]	Dominant	Postaxial polydactyly of hand
Lambotte Syndrome[3]	Recessive	Microcephaly, external auditory meatal atresia
Mesomelic Dysplasia[2] (bilateral aplasia of tibiae)	Recessive	Dwarfism, absence of thumbs, normal fibulae, aplastic tibiae
Oro-facio-digital Syndrome[2]		
Type I: Mohr-Claussen Syndrome	Recessive	Cleft palate, absent central incisors, bilateral hallucal polysyndactyly, brachydactyly, clinodactyly
Type IV[4]	Recessive	Tibial dysplasia, postaxial polydactyly, clubfoot, absent teeth, supernumerary teeth, cleft palate
Syndactyly-Polydactyly-Earlobe Syndrome[3]	Dominant	Postaxial polydactyly of hand
VATER association[2] (polydactyly/imperforate anus)	Dominant	Bifid vertebrae, hemivertebrae, sacral anomalies

[1] Inheritance is autosomal unless otherwise specified.
[2] Bergsma (1973).
[3] OMIM (1996).

lethalus and polydactyly with neonatal chondrodystrophy–type II).

Teratogenic agents are also implicated in the manifestation of preaxial polydactyly. Results from research by Graham et al. (1981) indicated that early compression of the embryo during weeks 4–6 could lead to necrosis in the limb bud mesoderm, resulting in preaxial polydactyly. Several studies (Martinez-Frias et al., 1992; Carey et al., 1990; Slee and Goldblatt, 1997) report a statistically significant relationship between mothers with nongestational diabetes and the presence of pedal preaxial polydactyly in the offspring.

PURPOSE OF THIS STUDY

The virtual absence of polydactyly in prehistory seems unlikely given the frequency with which it occurs in contemporary populations, particularly among many African populations. The susceptibility of small bones to postmortem damage, loss due to archaeological recovery techniques, and/or laboratory misidentification may be responsible for the limited examples of prehistoric polydactyly. To my knowledge, there is no reported prehistoric evidence for polydactyly in sub-Saharan Africa. This study describes the occurrence of polydactyly in an Iron Age skeleton from the site of Simbusenga in Zambia and considers the following: 1) how the case relates to clinical characterizations of polydactyly, 2) the possible etiology of the polydactyly in this case, and 3) comparative evidence for polydactyly in contemporary African populations.

DESCRIPTION OF SKELETON

The Iron Age site of Simbusenga (Fig. 2) is located in Zambia's Southern Province (Vogel, 1975). Radiocarbon dates from seven village horizons indicate occupation of the site from approximately AD 750–1575 (Vogel, 1975). Nine burials were excavated, but only eight were available for the study of paleopathology and dietary stable isotopes (Murphy, 1996). A single burial (Burial 1) may be intrusive and associated with modern radiocarbon dates, while the other eight burials are believed to date AD 1100–1500 on the basis of artifact associations (Vogel, 1975). I analyzed Burials 1–8 and identified two males, two females, and four adults of un-

Fig. 2. Map of Zambia showing the location of the archaeological site of Simbusenga.

known sex. Except for the modern burial, the skeletal remains are highly fragmentary. Other pathological conditions include dental caries, dental enamel hypoplasias, cribra orbitalia, trauma, and degenerative joint changes (Murphy, 1996).

Burial 5 is a young adult (14–25) with evidence of polydactyly. The radial head is fused, which can occur as early as 14 years of age. The iliac crest begins to fuse as early 17 years but is unfused in Burial 5 (based on McKern and Stewart, 1957). Third molar root development is complete in Burial 5, a process that usually occurs between 17 and 25 years (Hillson, 1996). The skeletal remains are both sparse and highly fragmentary, precluding sex determination.

The cranium is represented by the lateral portion of the left orbit and its zygomatic process. A total of 26 isolated permanent teeth are present. Postcranial remains are equally sparse. Long bone shafts and shaft fragments include the right humerus, left and right radii, ulnae, and right femur and tibia. The available material appears developmentally normal. Pelvic remains consist of right and left pubic rami (no symphyses) and the unfused right iliac crest epiphysis. Six rib head fragments are noted. The hands are represented by an incomplete right metacarpal (MC) II, incomplete MC IIIs, left MC IV, and twenty phalanges. All of the hand bones are normal in appearance. The feet are represented by both first metatarsals (MT I), an incomplete right MT II, and three pedal phalanges (none are first digit phalanges). Only the first MTs are anomalous in appearance. The only other pathological conditions associated with Burial 5 include a fissure pit caries on the buccal aspect of the LM_2 and dental enamel hypoplasias of all canines.

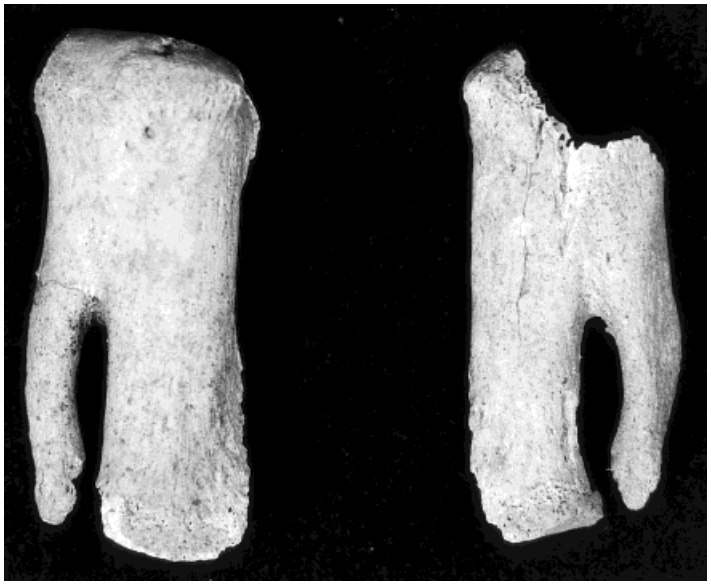

Fig. 3. Dorsal (superior) aspect of the left and right first metatarsals from Burial 5. Left metatarsal: 22.2 mm maximum width of head, 51.8 mm maximum length. Hypoplastic duplications originate from the metatarsal-phalangeal joint of each metatarsal and project proximally.

Both first metatarsals have evidence of incomplete (hypoplastic) digital duplication (Fig. 3). The left MT I shaft measures 51.8 mm, but postmortem damage prohibits shaft measurement of the right MT I. Both the shafts and the duplications are comprised of dense cortical bone. Each duplication emanates from the distal end of the lateral shaft and extends as a separate entity for 20.9 mm in the left MT I and 18.1 mm in the right MT I. The head of the left MT I is normal in appearance but broad, measuring 22.2 mm. The metatarsal-phalangeal joint of the left MT I may have two articular facets present, but the evidence is inconclusive due to postmortem damage in this area. Observations from the plantar view of both MT Is are also inhibited by extensive postmortem damage. The inferior tuberosity on the base of the left MT I is greatly reduced, due in part to the extension of the hypoplastic lateral duplication. The right MT1 sustained postmortem damage in this region. An inventory of hand and foot bones for the Simbusenga series (Table 2) indicates the absence of polydactyly in the other seven individuals.

TABLE 2. Inventory of hands and feet for the burials at Simbusenga

Burial number	Metacarpals I	II	III	IV	V	Phalanges	Metatarsals I	II	III	IV	V	Phalanges
Burial 1	2	2	2	1	1	8	1	—	1	1	1	5
Burial 2	1	2	2	1	1	12	1	1	1	1	1	—
Burial 3	—	—	1	1	—	—	—	—	—	—	—	—
Burial 4	1	—	—	—	—	2	2	—	2	—	1	1
Burial 5	—	1	2	1	—	20	2	—	—	—	1	3
Burial 6	2	—	1	1	1	7	—	1	1	—	1	—
Burial 7	1	2	2	2	2	22	2	2	1	2	2	7
Burial 8	2	2	2	1	1	16	—	—	—	—	—	—

DISCUSSION

Possible etiology of polydactyly in Burial 5

Morphologically, Burial 5's polydactyly is clinically classified as bilateral, preaxial polydactyly with hypoplastic lateral duplication of the first metatarsals (example 4, Fig. 1). Based on the previous discussion, there are three possible sources to explain the pedal preaxial polydactyly found in Burial 5: 1) an isolated occurrence, 2) manifestation of a syndrome, and 3) the result of a teratogenic agent.

Independent, or isolated, occurrences of preaxial polydactyly are the most common form of pedal polydactyly in population studies. For example, Venn-Watson (1976) reports that out of 72 individuals with polydactyly, only five of the patients were associated with a genetic syndrome. Studies of Utah birth records reveal that, from 1951–1961, 28 out of 59,561 infants had polydactyly, and of those with polydactyly only six of the 28 infants also had an associated syndrome (Woolf and Woolf, 1970).

Due to the incomplete nature of Burial 5's skeletal remains, it is not possible to rule out the presence of an associated syndrome; however, those syndromes with dental and limb abnormalities as well as syndromes with additional polydactyly are not likely responsible for the polydactyly seen in Burial 5 (Table 1). Burial 5 has relatively complete dentition that is normal in appearance. Lower limb abnormalities are associated with bilateral aplasia and Sakati syndromes, but Burial 5's lower limbs appear to be developmentally normal. Acrocallosal, Goiter et al., syndactyly-polydactyly-earlobe, and Curry-Jones syndromes are associated with additional polydactyly of the hands and feet. Although the hands and feet of Burial 5 are not complete (Table 2), the available elements show no evidence of additional polydactyly. Finally, it is impossible to evaluate the potential of four of the syndromes listed in Table 1, Noak, Carpenter, Lambotte, and polydactyly imperforate anus, due to the lack of diagnostic skeletal elements from Burial 5.

The possibility that the polydactyly in Burial 5 is the result of mechanical or disease-related teratogenesis, such as diabetes mellitus, cannot be ruled out. However, type II diabetes mellitus appears to be a modern phenomenon related to diets high in sugars and refined carbohydrates, decreasing activity levels, and increasing stress levels (Armstrong and McMichael, 1980; Nurse et al., 1985)—conditions not likely to have existed prehistorically at Simbusenga. Based on the evidence, this case is most likely an isolated incident independent of any associated syndrome or teratogenic process.

Polydactyly in Africa

Since high frequencies of polydactyly in populations of African ancestry are often cited in studies of population biology, cultural attitudes pertaining to polydactyly in Africa are of interest. Historically, childbirth laws among the Ibo of Nigeria indicate that infants born with deformities were killed, but "[in] the case of a six-fingered child the remedy is to amputate the finger; four fingers do not permit an equally easy remedy" (Thomas, 1913:60). Among Liberian groups, treatment of polydactyly ranges from amputation to acceptance (Schwab, 1974). The Wayao of Malawi believe that "A woman with polydactylism may not take food out of a corn store. Otherwise the stock of corn will not last long" (Stannus, 1922:307). Incidences of polydactyly vary between groups. For example, Bryant's (1949:114) ethnography of the Zulu indicates that "six-fingered and six-toed people occur, though rarely, among the Zulus, the superfluous limb (*umHlaza*) growing outside of the little finger or toe." Finally, Driberg's (1923) ethnography on the Lango of Uganda also claims polydactyly is rare but notes the occurrence of both preaxial and postaxial forms.

It is interesting that polydactyly is not afforded the same status in ethnographic accounts as it is in the human biology literature. In fact, most of the references are mentioned only incidentally in ethnographies discussing childbirth and the lives of children. While my ethnographic review was not exhaustive, the Human Relations Area Files contained only one additional reference to polydactyly. It is possible that polydactyly was not always viewed as an abnormality by other cultures. For example, among the Maasai, Merker (1910) states, "Excess fingers and toes are not amputated; they are not regarded as particularly unsightly."

Worldwide surveys in Table 3 suggest that the highest incidence of polydactyly occurs in Africans or in populations with African ancestry (Frazier, 1960; Sesgin and Stark, 1961; Scott-Emauakpor and Madueke, 1976; Simkiss and Lowe, 1961; Stevenson et al., 1966). However, Woolf and Myrianthopoulos (1973) argue that there are numerous types of polydactyly and that population frequencies vary by type. Nearly 100% of the polydactyly reported in African populations is postaxial in nature, and the majority of polydactylies in African-American groups are also postaxial, consisting of soft tissue structures (pendunculated postminimus) on the fifth digit (Scott-Emauakpor and Madueke, 1976; Simkiss and Lowe, 1961; Stevenson et al., 1966; Warkany, 1971). In contrast, in the Philippines, Hong Kong, and Ireland, 100% of polydactylies are preaxial in nature. The polydactyly present in Burial 5 is therefore unusual because of its extremely low frequency in African populations.

TABLE 3. The percentage of preaxial versus postaxial polydactyly among cases of polydactyly around the world

Country	% preaxial	% postaxial
Nigeria[5]	0	100
Uganda[6]	0	100
South Africa[7]	3	97
U.S. (Utah)[3]	59	34
U.S. (New Mexico)[2]	15	85
Mexico[7]	16	84
Brazil[7]	0	100
Uruguay, Chile, Argentina[4]	18	82
Chile[7]	14	86
Columbia[7]	14	86
Japan[1]	8	92
India[7]	14	86
Australia[8]	33	66
Spain[7]	50	50
Yugoslavia[7]	60	40
Czechoslovakia[7]	40	60
Ireland[7]	100	0
Hong Kong[7]	100	0
Malaysia[7]	88	12
Philippines[7]	100	0

[1] Watanabe et al. (1992).
[2] Phelps and Grogan (1985).
[3] Woolf and Woolf (1970).
[4] Castilla et al. (1973).
[5] Scott-Emuakpor and Madueke (1976).
[6] Simkiss and Lowe (1961).
[7] Stevenson et al. (1966).

CONCLUSIONS

Based on the clinical literature, the digital abnormality in Burial 5 from the site of Simbusenga, Zambia, is identified as bilateral, preaxial polydactyly of the first metatarsal. The exact etiology of the polydactyly is unknown, due in part to the fragmentary nature of the specimen. While syndromes and teratogenic processes cannot be ruled out, this case is most likely the result of an isolated genetic phenomenon.

African and African-American groups have a high frequency of postaxial polydactyly. Preaxial polydactyly is still a rare occurrence in these populations, making this case of prehistoric polydactyly even more significant. Finally, the occurrence of polydactyly in this study and others (Barnes, 1994; Hill and Case, 1996) indicates that, with careful recovery and analysis, it is possible to find examples of these skeletal abnormalities in prehistoric remains.

ACKNOWLEDGMENTS

I thank the staff at the Livingstone Museum and National Monuments in Zambia for access to skeletal material. Editing comments by Paul Jamison, Della Cook, Bruce Hardy, and "the editing group" were greatly appreciated. Finally, I acknowledge Bruce Hardy's assistance with the figures. This work was funded by a Fulbright IIE grant.

LITERATURE CITED

Armstrong BK, McMichael AJ. 1980. Overnutrition. In: Stanley NF, Joske RA, editors. Changing disease patterns and human behaviour. Academic Press: London. p 491–506.

Barnes E. 1994. Polydactyly in the Southwest. Kiva 59:419–431.

Bergsma D. 1973. Birth defects: atlas and compendium. Baltimore: Williams and Wilkins Co.

Bryant AT. 1949. The Zulu people as they were before the white man came. Pietermaritzburg: Shuter and Shooter.

Carey JD, Hommell M, Fineman RM, Hall BD. 1990. Hallucal polydactyly in infants of diabetic mothers: a clinical marker and possible clue to teratogenesis. Proceedings of the Greenwood Genetic Center 9:95.

Castilla E, Paz J, Mutchinick O, Munoz E, Giorgiutti E, Gelman Z. 1973. Polydactyly: a genetic study in South America. Am J Hum Genet 25:405–412.

Christensen JC, Leff FB, Lepow GM, Schwartz RI, Colon PA, Arminio ST, Nixon P, Segel D, Leff S. 1981. Congenital polydactyly and polymetatarsalia: classification genetics and surgical correction. J Foot Surg 20:151–158.

Chung CS, Myrianthopoulos NC. 1968. Racial and prenatal factors in major congenital malformations. Am J Hum Genet 20:44–60.

Cross TP. 1952. Motif index of 1952. Folklore series 7. Bloomington, IN: Indiana University Press.

Dooley JL, Niehaus LP. 1985. Preaxial polydactyly. J Foot Surg 24:122–126.

Driberg JH. 1923. The Lango. A Nilotic tribe of Uganda. London: T. Fisher Unwin.

Frazier TM. 1960. A note on race-specific congenital malformation rates. Am J Obstet Gynecol 80:184–185.

Graham JM, Higginbottom MC, Smith DW. 1981. Preaxial polydactyly of the foot associated with early amnion rupture: evidence for mechanical teratogenesis? J Pediatr 98:943–945.

Hennekam RCM. 1991. Short rib syndrome-Beemer type in sibs. Am J Med Genet 40:230–233.

Hill RJ, Case DT. 1996. Bilateral postaxial polydactyly of the foot in a Salado infant. In: Cockburn E, editor. Papers on Paleopathology Presented at the Twenty-third Annual Meeting of the Paleopathology Association, Durham, North Carolina, 9–10 April 1996. Detroit, MI: Paleopathology Association. p. 18 [poster].

Hillson S. 1996. Dental anthropology. Cambridge: Cambridge University Press.

Martinez-Frias ML, Bermejo E, Cereijo A. 1992. Preaxial polydactyly of feet in infants of diabetic mothers: epidemiological test of a clinical hypothesis. Am J Med Genet 42:643–646.

Masada K, Tsuyuguchi Y, Kawabata H, Ono K. 1987. Treatment of preaxial polydactyly of the foot. Plast Reconstr Surg 79:251–258.

McKern TW, Stewart TD. 1957. Skeletal age changes in young American males. Technical report EP-45. Natick, MA: Quartermaster Research and Development Command.

Merker M. 1910. Die Masai: Ethnographische monographie eines Ostafrikanischen Semitenvolkes. Berlin: Dietrich Reimer (Ernst Vohsen).

Murphy KA. 1996. The skeletal elements of the Iron Age in central and southern Africa: a bioarchaeological approach to the reconstruction of prehistoric subsistence. PhD thesis, Indiana University.
Nicolai J-PA, Schoch SL. 1986. Polydactyly in the Bible. J Hand Surg 11a:293.
Nurse GT, Weiner JS, Jenkins T. 1985. The peoples of Southern Africa and their affinities. Oxford: Clarendon Press.
OMIM™ (Online Mendelian Inheritance in Man). 1996. Center for Medical Genetics, Johns Hopkins University (Baltimore, MD) and National Center for Biotechnology Information,
National Library of Medicine (Bethesda, MD). World Wide Web URL: http://www.ncbi.nlm.mih.gov/Omim/
Phelps DA, Grogan DP. 1985. Polydactyly of the foot. J Pediatr Orthop 5:446–451.
Radhakrishna U, Multani AS, Solanki JV, Shah VC, Chinoy NJ. 1993. Polydactyly: a study of a five generation Indian family. J Med Genet 30:296–299.
Schwab G. 1974. Tribes of the Liberian hinterland. New York: Kraus Reprint Co.
Scott-Emuakpor AB, Madueke E-DN. 1976. The study of genetic variation in Nigeria. II. The genetics of polydactyly. Hum Hered 26:198–202.
Sesgin MD, Stark RB. 1961. The incidence of congenital defects. J Plast Reconstr Surg 27:261–267.
Simkiss M, Lowe A. 1961. Congenital abnormalities in the African newborn. Arch Dis Child 36:404–406.
Slee J, Goldblatt J. 1997. Further evidence for preaxial hallucal polydactyly as a marker of diabetic embryopathy. J Med Genet 34:261–263.
Sonoga AL, Guttmann GG. 1989. Pedal polydactyly. J Am Podiatr Med Assoc 9:254–258.
Stannus HS. 1922. The Wayao of Nyasaland. Harvard Afr. Studies 3:229–372.
Stevenson AC, Johnston HA, Stewart MIP, Golding DR. 1966. Congenital malformations. Geneva: WHO.
Temtamy S, McKusick VA. 1969. The genetics of hand malformations with particular emphasis on genetic factors. Birth Defects 14:364–423.
Thomas NW. 1913. Anthropological report on the Ibo-speaking peoples of Nigeria. Part I. Law and custom of the Ibo of the Awaka neighbourhood, southern Nigeria. London: Harrison and Sons.
Venn-Watson EA. 1976. Problems in polydactyly of the foot. Orthop Clin North Am 7:909–927.
Vogel JO. 1975. Simbusenga. Zambia museums papers 4. London: Oxford University Press.
Walker JT. 1961. A pedigree of extra-digit V polydactyly in a Batutsi family. Ann Hum Genet 25:65–68.
Warkany J. 1971. Congenital malformations. Notes and comments. Chicago: Year Book Medical Publishers.
Watanabe H, Fujita S, Oka I. 1992. Polydactyly of the foot: an analysis of 265 cases and a morphological classification. Plast Reconstr Surg 89:856–877.
Weir A. 1991. The six wives of Henry VIII. New York: Ballentine Books.
Woolf CM, Myrianthopoulos MC. 1973. Polydactyly in American Negroes and whites. Hum Genet 25:397–404.
Woolf CM, Woolf RM. 1970. A genetic study of polydactyly in Utah. Hum Genet 22:75–88.